BRIEF ARTICLE

Primary Cutaneous Follicle Center Lymphoma in an Adult African American Male

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ABSTRACT

Primary cutaneous B-Cell Lymphomas (PCBCL) is a form of a lymphoproliferative neoplasm consisting of proliferation localized to the skin. Cutaneous lymphomas originate from mature T-lymphocytes, B-lymphocytes, or NK cells. The incidence of PCBCL is significantly lower in patients with Skin of Color (SoC) and is quite rare among African American (AA) individuals, compared to their Caucasian counterparts. The clinical course of PCFCL in SoC is not widely studied or reported in literature due to the low incidence in the AA population. We present a case of PCBCL in an AA individual along with a brief review of the clinical presentation, pathology, and treatment options of the condition. According to the U.S. Census Bureau, approximately half of the U.S population will have SoC by 2050; by adding our case of PCFCL to the body of literature, we hope to promote awareness of the presentation and course of this cutaneous disorder in SoC patients.

INTRODUCTION

Primarv cutaneous lymphomas are lymphoproliferative neoplasms consisting of proliferation localized to the skin. Cutaneous lymphomas (CLs) originate from mature Tlymphocytes, B-lymphocytes, or NK cells.¹ During a study of the National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) database for the years 2004–2008, the incident rates for Primary cutaneous B-Cell Lymphomas (PCBCL) in white persons and African American (AA) persons were reported as 3.6 per 1 million and 1.8 per 1 million, respectively.¹ This supports current notions in literature that the incidence of PCBCL in AA is very low.

PCBCL can be further subdivided according to the WHO-European Organization for Research and Treatment of Cancer (WHO-EORTC), into three types: primarv cutaneous marginal zone lymphoma, primary follicle center Ivmphoma cutaneous (PCFCL), and primary cutaneous diffuse B-cell large lymphoma.² Cutaneous pathology can be more challenging to recognize in patients with Skin of Color (SoC) and PCFCL is quite rare among AA individuals. We discuss the clinical findings of an AA patient diagnosed with PCFCL in hopes of bringing awareness to the varying morphologic presentations of PCBCL.

CASE REPORT

A 61-year-old AA gentleman, Fitzpatrick VI, presented with a 1.5 cm X 1.5 cm asymptomatic, firm, dermal, hyperpigmented nodule located on his right upper forehead, growing in size (Figure 1). There was no surrounding erythema, tenderness, or scale. Histopathology showed a superficial and deep periadnexal and interstitial lymphoid infiltrate extending into the subcutaneous fat associated with stromal fibrosis (Figure 2a). Germinal centers were identified in the deep reticular dermis. Immunohistochemical staining revealed CD20-positive B-cells predominating over reactive CD3 and CD5positive T-cells (Figure 2b). Molecular studies demonstrated positive PCR for immunoglobulin heavy chain aene rearrangements, confirming the presence of a clonal B-cell population. The patient was diagnosed with PCFCL. Work-up included CMP, CBC, HIV, Hepatitis panel, flow cytometry, and whole-body PET/CT, which were noncontributory. Due to patient preference, a three-month trial of topical betamethasone 0.05% ointment twice daily was initiated without improvement. Since his initial presentation, the patient's lesions have remained unchanged without additional treatment, as he has declined further intervention at this time.

DISCUSSION

PCFCL typically presents with slow-growing, erythematous, painless, solitary nodules or plaques smaller than 5.0 cm, classically located on the head and neck. One study found that the anatomic distribution among five AA patients had lesions distributed in the classic locations, while white participants mostly presented with solitary lesions that were more widely distributed anatomically. ^{3,4}

Histologically, PCFCL demonstrates an epidermis-sparing B-cell infiltrate comprised

of centrocytes and centroblasts with a follicular or diffuse pattern.⁵ B-cell lineage is confirmed by the presence of CD20 or CD79a and the absence of CD3.⁴ Due to its rarity, current large studies regarding treatment consensus are lacking but the typically indolent nature of PCFCL has led to localized, low morbidity modalities such as radiotherapy or surgical removal as the mainstays of therapy.³ Given the large size and location of many of these lesions, surgical excision may not be cosmetically favorable, especially in darker skin tones prone to post-inflammatory hyperpigmentation and increased scarring.

Limited data exists regarding the use of both topical and intralesional corticosteroids for the treatment of PCFCL. A literature review revealed a single case series in which three patients were treated with intralesional corticosteroids (ILC) for PCFCL, all of whom saw a beneficial response. In a separate but similar case study a participant received topical steroids and ILC that resulted in regression of the lesion after failed therapy with electron beam.⁶

CONCLUSION

The clinical course of PCFCL in SoC is not widely studied or reported in literature due to the low incidence in the AA population. Dermatologists are becomina more cognizant of the fact that both common and rare cutaneous diseases in patients with SoC lack representation in literature and educational texts. Adding our case of PCFCL to the body of literature will promote awareness of the presentation and course of this cutaneous disorder in SoC patients.

Conflict of Interest Disclosures: None

Funding: None

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Figure 1. Hyperpigmented nodule located on the right upper forehead.



Figure 2. A) Histopathology of hyperpigmented nodule. B) Immunohistochemical staining.